

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:sssptal635jxs

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

\* \* \* \* \* Welcome to STN International \* \* \* \* \*

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America  
NEWS 2 "Ask CAS" for self-help around the clock  
NEWS 3 May 10 PROUSDDR now available on STN  
NEWS 4 May 19 PROUSDDR: One FREE connect hour, per account, in both May  
and June 2004  
NEWS 5 May 12 EXTEND option available in structure searching  
NEWS 6 May 12 Polymer links for the POLYLINK command completed in REGISTRY  
NEWS 7 May 17 FRFULL now available on STN  
NEWS 8 May 27 New UPM (Update Code Maximum) field for more efficient patent  
SDIs in Cplus  
NEWS 9 May 27 Cplus super roles and document types searchable in REGISTRY  
NEWS 10 May 27 Explore APOLLIT with free connect time in June 2004  
NEWS 11 Jun 22 STN Patent Forums to be held July 19-22, 2004

NEWS EXPRESS MARCH 31 CURRENT WINDOWS VERSION IS V7.00A, CURRENT  
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),  
AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004  
NEWS HOURS STN Operating Hours Plus Help Desk Availability  
NEWS INTER General Internet Information  
NEWS LOGIN Welcome Banner and News Items  
NEWS PHONE Direct Dial and Telecommunication Network Access to STN  
NEWS WWW CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that  
specific topic.

All use of STN is subject to the provisions of the STN Customer  
agreement. Please note that this agreement limits use to scientific  
research. Use for software development or design or implementation  
of commercial gateways or other similar uses is prohibited and may  
result in loss of user privileges and other penalties.

\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 15:17:55 ON 22 JUN 2004

=> FIL MEDLINE BIOSIS EMBASE CA SCISEARCH

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 15:18:01 ON 22 JUN 2004

FILE 'BIOSIS' ENTERED AT 15:18:01 ON 22 JUN 2004

COPYRIGHT (C) 2004 BIOLOGICAL ABSTRACTS INC. (R)

FILE 'EMBASE' ENTERED AT 15:18:01 ON 22 JUN 2004

COPYRIGHT (C) 2004 Elsevier Inc. All rights reserved.

FILE 'CA' ENTERED AT 15:18:01 ON 22 JUN 2004  
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.  
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.  
COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'SCISEARCH' ENTERED AT 15:18:01 ON 22 JUN 2004  
COPYRIGHT 2004 THOMSON ISI

=> s (Ship (n) 1) or (SH2 (2n) phosphatidylinositol phosphatase) or Ship or INPP5D  
L1 32068 (SHIP (N) 1) OR (SH2 (2N) PHOSPHATIDYLINOSITOL PHOSPHATASE) OR  
SHIP OR INPP5D

=> s antisense or (anti (n) sense) or (complemen (n2) (oligonucl? or nucl?))  
MISSING OPERATOR 'COMPLEMEN (N2)'

The search profile that was entered contains terms or  
nested terms that are not separated by a logical operator.

=> s antisense or (anti (n) sense) or (complemen (2n) (oligonucl? or nucl?))  
L2 120052 ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMEN (2N) (OLIGONUCL? OR  
NUCL?))

=> s antisense or (anti (n) sense) or (complemen? (2n) (oligonucl? or nucl?))  
L3 140133 ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMEN? (2N) (OLIGONUCL?  
OR NUCL?))

=> s l1 and l3  
L4 26 L1 AND L3

=> dup rem l4  
PROCESSING COMPLETED FOR L4  
L5 13 DUP REM L4 (13 DUPLICATES REMOVED)

=> s l5 py<=2000  
MISSING OPERATOR L5 PY<=2000  
The search profile that was entered contains terms or  
nested terms that are not separated by a logical operator.

=> s l5 and py<=2000  
2 FILES SEARCHED...  
L6 3 L5 AND PY<=2000

=> d l6 ibib abs 1-6

L6 ANSWER 1 OF 3 MEDLINE on STN  
ACCESSION NUMBER: 2000459945 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 10958682  
TITLE: 5' phospholipid phosphatase **SHIP**-2 causes protein  
kinase B inactivation and cell cycle arrest in glioblastoma  
cells.  
AUTHOR: Taylor V; Wong M; Brandts C; Reilly L; Dean N M; Cowser L  
M; Moodie S; Stokoe D  
CORPORATE SOURCE: Cancer Research Institute, University of California, San  
Francisco 94115, USA.  
CONTRACT NUMBER: R01CA79548 (NCI)  
SOURCE: Molecular and cellular biology, (2000 Sep) 20  
(18) 6860-71.  
Journal code: 8109087. ISSN: 0270-7306.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200009  
ENTRY DATE: Entered STN: 20001005

Last Updated on STN: 20001005

Entered Medline: 20000922

AB The tumor suppressor protein PTEN is mutated in glioblastoma multiform brain tumors, resulting in deregulated signaling through the phosphoinositide 3-kinase (PI3K)-protein kinase B (PKB) pathway, which is critical for maintaining proliferation and survival. We have examined the relative roles of the two major phospholipid products of PI3K activity, phosphatidylinositol 3,4-bisphosphate [PtdIns(3,4)P<sub>2</sub>] and phosphatidylinositol 3,4,5-trisphosphate [PtdIns(3,4,5)P<sub>3</sub>], in the regulation of PKB activity in glioblastoma cells containing high levels of both of these lipids due to defective PTEN expression. Reexpression of PTEN or treatment with the PI3K inhibitor LY294002 abolished the levels of both PtdIns(3, 4)P<sub>2</sub> and PtdIns(3,4,5)P<sub>3</sub>, reduced phosphorylation of PKB on Thr308 and Ser473, and inhibited PKB activity. Overexpression of **SHIP-2** abolished the levels of PtdIns(3,4,5)P<sub>3</sub>, whereas PtdIns(3,4)P<sub>2</sub> levels remained high. However, PKB phosphorylation and activity were reduced to the same extent as they were with PTEN expression. PTEN and **SHIP-2** also significantly decreased the amount of PKB associated with cell membranes. Reduction of **SHIP-2** levels using **antisense** oligonucleotides increased PKB activity. **SHIP-2** became tyrosine phosphorylated following stimulation by growth factors, but this did not significantly alter its phosphatase activity or ability to antagonize PKB activation. Finally we found that **SHIP-2**, like PTEN, caused a potent cell cycle arrest in G(1) in glioblastoma cells, which is associated with an increase in the stability of expression of the cell cycle inhibitor p27(KIP1). Our results suggest that **SHIP-2** plays a negative role in regulating the PI3K-PKB pathway.

L6 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
ACCESSION NUMBER: 2000:360149 BIOSIS  
DOCUMENT NUMBER: PREV200000360149  
TITLE: **Antisense** modulation of **Ship-2** expression.  
AUTHOR(S): Bennett, C. Frank [Inventor]; Cowser, Lex M. [Inventor]  
CORPORATE SOURCE: ASSIGNEE: Isis Pharmaceuticals Inc.  
PATENT INFORMATION: US 6025198 February 15, 2000  
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Feb. 15, 2000) Vol. 1231, No. 3. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 23 Aug 2000  
Last Updated on STN: 8 Jan 2002

AB **Antisense** compounds, compositions and methods are provided for modulating the expression of **Ship-2**. The compositions comprise **antisense** compounds, particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **Ship-2**. Methods of using these compounds for modulation of **Ship-2** expression and for treatment of diseases associated with expression of **Ship-2** are provided.

L6 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
ACCESSION NUMBER: 1998:44397 BIOSIS  
DOCUMENT NUMBER: PREV199800044397  
TITLE: Keratin 8 and 18 expression in mesenchymal progenitor cells of regenerating limbs is associated with cell proliferation and differentiation.  
AUTHOR(S): Corcoran, Jonathan P.; Ferretti, Patrizia [Reprint author]  
CORPORATE SOURCE: Dev. Biol. Unit, Inst. Child Health, UCL, 30 Guilford St., London WC1N 1EH, UK  
SOURCE: Developmental Dynamics, (Dec., 1997) Vol. 210, No. 4, pp. 355-370. print.  
CODEN: DEDYEI. ISSN: 1058-8388.

DOCUMENT TYPE: Article  
LANGUAGE: English  
OTHER SOURCE: Genbank-136454  
ENTRY DATE: Entered STN: 27 Jan 1998  
Last Updated on STN: 27 Jan 1998

AB Keratins are considered markers of epithelial differentiation. In lower vertebrates, however, immunoreactivity for keratin 8 and 18 has been reported in nonepithelial cells, particularly in mesenchymal progenitor cells of regenerating complex body structures. To confirm that such reactivity does indeed reflect keratin expression and to investigate their possible role in regeneration, we have isolated clones coding for the new homologues of keratin 8 and 18 (NvK8 and NvK18, respectively) and studied their distribution and changes in their expression following experimental manipulations. Analysis of NvK8 and NvK18 transcripts confirms that KS and K18 are expressed in the blastemal cells of regenerating newt limbs and that their expression is first observed 3-5 days after amputation, when the blastemal cells start to proliferate under the influence of the nerve, whose presence is essential for regeneration to proceed. In contrast, no induction of these keratins is observed following amputation of a larval limb at a stage when organogenesis is proceeding in a nerve-independent manner. To establish whether there is a causal relation. **ship** between keratin expression and cell proliferation in the adult limb blastema, we have investigated whether their expression is nerve-dependent and whether suppression of their expression in cultured blastemal cells affects cell division and differentiation. Analysis of keratins in denervated limbs demonstrates that the nerve is not necessary to induce their expression. However, treatment of cultured blastemal cells with KS and K18 **anti-sense** oligonucleotides significantly decreases DNA synthesis and induces changes in cell morphology, suggesting that expression of these keratins during regeneration may be necessary for the maintenance of the undifferentiated and proliferative state of blastemal cells.

=> d his

(FILE 'HOME' ENTERED AT 15:17:55 ON 22 JUN 2004)

FILE 'MEDLINE, BIOSIS, EMBASE, CA, SCISEARCH' ENTERED AT 15:18:01 ON 22 JUN 2004

L1 32068 S (SHIP (N) 1) OR (SH2 (2N) PHOSPHATIDYLINOSITOL PHOSPHATASE) O  
L2 120052 S ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMEN (2N) (OLIGONUCL?  
L3 140133 S ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMEN? (2N) (OLIGONUCL?  
L4 26 S L1 AND L3  
L5 13 DUP REM L4 (13 DUPLICATES REMOVED)  
L6 3 S L5 AND PY<=2000

=> s l4 and (l3 (w) l1)

L7 0 L4 AND (L3 (W) L1)

=> d l5 ibib abs 1-13

L5 ANSWER 1 OF 13 CA COPYRIGHT 2004 ACS on STN  
ACCESSION NUMBER: 140:350580 CA  
TITLE: Method for inhibiting angiogenesis with **Ship**  
-1 inhibitors  
INVENTOR(S): Marcusson, Eric G.; Dean, Nicholas M.  
PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., USA  
SOURCE: PCT Int. Appl., 26 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004032880	A2	20040422	WO 2003-US32494	20031014
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: US 2002-418393P P 20021011

AB Methods for inhibiting angiogenesis using inhibitors of **Ship-1** are provided. The net result is prevention, reduction or treatment of angiogenesis. Methods of treating angiogenic diseases and conditions and conditions associated with aberrant or excessive blood vessel growth are provided. **Ship-1** inhibitors of the invention include small mols., antibodies, peptides (including dominant neg. peptides) and **antisense** compds., including ribozymes, inhibitory RNA mols. including siRNA mols. and **antisense** oligonucleotides.

L5 ANSWER 2 OF 13 CA COPYRIGHT 2004 ACS on STN  
 ACCESSION NUMBER: 139:333972 CA  
 TITLE: Gene profiling methods of diagnosing potential for metastasis or developing hepatocellular carcinoma and of identifying therapeutic targets  
 INVENTOR(S): Wang, Xin Wei; Ye, Qing-hai; Kim, Jin Woo  
 PATENT ASSIGNEE(S): The Government of the United States of America, as Represented by the Secretary of the Department of Health and Human Services, USA  
 SOURCE: PCT Int. Appl., 141 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003087766	A2	20031023	WO 2003-US10783	20030404
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: US 2002-370895P P 20020405

AB The present invention relates to methods for diagnosing the metastatic potential of hepatocellular carcinoma (HCC) in HCC patients and methods for diagnosing the potential of developing HCC in patients with chronic liver diseases. A computer readable medium, a digital computer, and a system useful for such diagnosis are also provided. Further disclosed are methods for identifying potential therapeutic targets for treating metastasis in HCC patients and methods for preventing HCC in patients with chronic liver diseases. Based on UniGene (UG) database compiled by NCBI,

two sets of gene clusters: Metastatic gene expression predictor correlated with the diagnosis of metastatic HCC and HCC gene expression predictor correlated with the diagnosis of patients likely to develop HCC, are identified by gene profiling method. Among them, osteopontin (OPN) and EpCAM (Epithelial Cell Adhesion Mol., also known as TACSTD1, encoded by gene GA733-2) are used as the major therapeutic targets (both sequences claimed but not provided). In addition, the invention provides methods for inhibiting metastasis in HCC patients by suppressing the function of one therapeutic target, osteopontin, and methods for preventing the development of HCC in patients with chronic liver diseases by suppressing the function of one therapeutic target, EpCAM. Pharmaceutical compns. containing agents capable of inhibiting the functions of osteopontin or EpCAM are also disclosed.

L5 ANSWER 3 OF 13 CA COPYRIGHT 2004 ACS on STN  
 ACCESSION NUMBER: 139:95435 CA  
 TITLE: Modified receptors on cell membranes for the discovery of therapeutic ligands  
 INVENTOR(S): Schwartz, Thue W.; Martini, Lene; Heydorn, Arne; Jorgensen, Rasmus  
 PATENT ASSIGNEE(S): 7TM Pharma A/S, Den.  
 SOURCE: PCT Int. Appl., 122 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 2  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003055914	A2	20030710	WO 2002-DK900	20021220
WO 2003055914	A3	20031023		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DK 2001-1944 A 20011221  
 DK 2002-113 A 20020122  
 DK 2002-1043 A 20020703  
 US 2002-394122P P 20020703

AB A drug discovery method is provided for selecting a compound selected from the group consisting of a small organic substance, a biopharmaceutical, or an antibody or part thereof. The method comprises the steps of (i) expressing one or more receptors on a cell membrane, such as, e.g., an exterior cell surface of a cell, (ii) contacting one or more expressed receptors with a test compound or a selection of test compds. (libraries), and (iii) selecting one or more compds. based on its ability to bind one or more receptors. The step of expressing the one or more receptors comprises capturing one or more receptors on the exterior cell surface in a conformation that predominantly enables binding or interaction with a ligand, and the conformation that predominantly enables binding or interaction with a ligand is provided by modification of one or more receptors by a method comprising at least one of the following: (a) fusion with any protein which keeps the receptor in the desired conformation such as, e.g. an arrestin, a modified arrestin, a G-protein or a modified G-protein, (b) site-directed mutagenesis, and (c) deletion. The receptors may be captured on the exterior cell surface by at least one of the

following: (d) interaction of the receptor with a scaffolding protein, optionally, with a scaffolding protein network and (e) means for blocking receptor internalization, e.g. by co-expression of a mutated dynamin or a modified arrestin or by use of chems. such as, e.g., sucrose and/or Tris. Thus, by coexpressing of either the wild-type receptor or by modifying the receptor by engineering for example a recognition motif for a strong binder into its structure (for example, a PDZ recognition motif at its C-terminal end), and coexpression of this with a scaffolding protein such as PSD-95 or a modified scaffolding protein which interacts with the cytoskeleton at the cell surface or is made to be closely associated with the membrane through a lipid anchor, a high level of surface expression can be ensured, which will benefit its use in the drug discovery process. As a result of the strong tendency of the scaffolding proteins to interact with each other, just the cotransfection with one or more appropriate scaffolding proteins or modified scaffolding protein may also lead to the formation of patches with high local concns of the receptor or modified receptor, which will be highly beneficial in the drug discovery process where they are used initially to select binding mols. The method is exemplified by expression of the NK1 receptor in an agonist high-affinity binding form at the surface of transfected cells through fusion with arrestin or the N-terminal fragment of arrestin.

L5 ANSWER 4 OF 13 CA COPYRIGHT 2004 ACS on STN  
 ACCESSION NUMBER: 139:47146 CA  
 TITLE: **Antisense** modulation of SH2-containing inositol 5-phosphatase (**SHIP-1**) expression for treatment of inflammatory disorders  
 INVENTOR(S): Bennett, C. Frank; Freier, Susan M.  
 PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., USA  
 SOURCE: U.S. Pat. Appl. Publ., 46 pp.  
 CODEN: USXXCO  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003114401	A1	20030619	US 2001-3919	20011206
WO 2003053341	A2	20030703	WO 2002-US38622	20021204

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM  
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-3919 A 20011206

AB **Antisense** compds., compns. and methods are provided for modulating the expression of **Ship-1**. The compns. comprise **antisense** compds., particularly **antisense** oligonucleotides, targeted to nucleic acids encoding **Ship-1**. Methods of using these compds. for modulation of **Ship-1** expression and for treatment of diseases associated with expression of **Ship-1** are provided.

L5 ANSWER 5 OF 13 CA COPYRIGHT 2004 ACS on STN  
 ACCESSION NUMBER: 136:257243 CA  
 TITLE: Control of NK cell function and survival by modulation of **SHIP** activity  
 INVENTOR(S): Kerr, William G.

PATENT ASSIGNEE(S): University of South Florida, USA  
SOURCE: PCT Int. Appl., 49 pp.  
CODEN: PIXXD2  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 2  
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002024233	A2	20020328	WO 2001-US29158	20010919
WO 2002024233	A3	20030313		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
AU 2001092753	A5	20020402	AU 2001-92753	20010919
EP 1318841	A2	20030618	EP 2001-973144	20010919
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR			

PRIORITY APPLN. INFO.:  
US 2000-233661P P 20000919  
US 2001-314099P P 20010823  
WO 2001-US29158 W 20010919

AB Suppression of hematopoietic-specific SH2-containing inositol polyphosphatase (**SHIP**) activity by genetic and pharmaceutical means is taught for suppression of rejection of, and prevention of graft-vs.-host disease in, solid organ allografts or xenotransplants, and histo-incompatible marrow grafts. Also disclosed are methods for the screening of substances and genetic constructs that inhibit **SHIP** function in mammalian cells, and cell lines and transgenic animals that have the **SHIP** -/- phenotype.

L5 ANSWER 6 OF 13 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2002400446 MEDLINE

DOCUMENT NUMBER: PubMed ID: 12149650

TITLE: PTEN, but not **SHIP** and SHIP2, suppresses the PI3K/Akt pathway and induces growth inhibition and apoptosis of myeloma cells.

AUTHOR: Choi Yong; Zhang Jie; Murga Cristina; Yu Hong; Koller Erich; Monia Brett P; Gutkind J Silvio; Li Weiqun

CORPORATE SOURCE: Lomabardi Cancer Center, Georgetown University Medical Center, Washington, District of Colombia 20007, USA.

SOURCE: Oncogene, (2002 Aug 8) 21 (34) 5289-300.  
Journal code: 8711562. ISSN: 0950-9232.

PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200208

ENTRY DATE: Entered STN: 20020801

Last Updated on STN: 20020831

Entered Medline: 20020830

AB Expression of PTEN tumor suppressor gene has been known to dephosphorylate the phosphatidylinositol 3' kinase (PI3K) products on the 3 prime inositol ring, resulting in reduced Akt activation. Loss of PTEN expression in OPM2 and delta47 human myeloma lines led to high Akt activity toward insulin-like growth factor I (IGF-I). In contrast, mouse plasma cell tumor (PCT) lines, expressing wild type PTEN, did not respond to IGF-I for Akt activation. We demonstrated here that endogenous PTEN played a

negative role in controlling Akt activity in both mouse PCT and NIH3T3 fibroblast lines by using **anti-sense** oligonucleotides against PTEN. To determine the role of src-homology 2-containing inositol 5' phosphatase (**SHIP**) in regulating the PI3K/Akt pathway, we manipulated its expression by down-regulation and overexpression in myeloma, PCT and NIH3T3 lines and analysed Akt activation. Our results showed that **SHIP**, unlike PTEN, did not affect Akt activity in all systems analysed, despite its ability to dephosphorylate a PI3K product. Although SHIP2 expression resulted in suppression of interleukin-6-mediated mitogen-activated protein kinase activation, expression of **SHIP** and SHIP2 in a PTEN-null myeloma line did not suppress Akt activity. Biologically, expression of only PTEN, but not **SHIP** and SHIP2, resulted in growth inhibition and increased apoptosis in OPM2 myeloma line. Together, our results have established the role of PTEN, but not **SHIP** and SHIP2, in negatively regulating the PI3K/Akt cascade and in myeloma leukemogenesis.

L5 ANSWER 7 OF 13 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN  
 ACCESSION NUMBER: 2002:840149 SCISEARCH  
 THE GENUINE ARTICLE: 598GC  
 TITLE: Membrane localization of Src homology 2-containing inositol 5'-phosphatase 2 via Shc association is required for the negative regulation of insulin signaling in Rat1 fibroblasts overexpressing insulin receptors  
 AUTHOR: Ishihara H; Sasaoka T (Reprint); Ishiki M; Wada T; Hori H; Kagawa S; Kobayashi M  
 CORPORATE SOURCE: Toyama Med & Pharmaceut Univ, Dept Clin Pharmacol, 2630 Sugitani, Toyama 9300194, Japan (Reprint); Toyama Med & Pharmaceut Univ, Dept Clin Pharmacol, Toyama 9300194, Japan; Toyama Med & Pharmaceut Univ, Dept Internal Med 1, Toyama 9300194, Japan; Sainou Hosp, Toyama 9300887, Japan  
 COUNTRY OF AUTHOR: Japan  
 SOURCE: MOLECULAR ENDOCRINOLOGY, (OCT 2002) Vol. 16, No. 10, pp. 2371-2381.  
 Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE 500, BETHESDA, MD 20814-4110 USA.  
 ISSN: 0888-8809.  
 DOCUMENT TYPE: Article; Journal  
 LANGUAGE: English  
 REFERENCE COUNT: 40

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB Lipid phosphatase SHIP2 [Src homology 2 (SH2)-containing inositol 5'-phosphatase 2] has been shown to be a physiologically critical negative regulator of insulin signaling. We investigated the molecular mechanism by which SHIP2 negatively regulates insulin-induced phosphorylation of Akt, a key downstream molecule of phosphatidylinositol 3-kinase important for the biological action of insulin. Overexpression of wild-type SHIP2 (WT-SHIP2) inhibited insulin-induced phosphorylation of Akt at both Thr(308) and Ser(473) in Rat1 fibroblasts expressing insulin receptors. The degree of inhibition was less in the cells expressing either a mutant SHIP2 with R47Q change (R/Q-SHIP2) in the SH2 domain, or a mutant SHIP2 with Y987F change (Y/F-SHIP2) in the C-terminal tyrosine phosphorylation site. However, on addition of a myristoylation signal, WT-SHIP2, R/Q-SHIP2, and Y/F-SHIP2 all efficiently inhibited insulin-induced Akt phosphorylation at both residues, whereas a 5'-phosphatase-defective mutant SHIP2 (DeltaIP-SHIP2) with the myristoylation signal did not. Interestingly, the degree of inhibition of Akt phosphorylation by R/Q-SHIP2 and Y/F-SHIP2 is well correlated with the extent of their association with Shc. In addition, overexpression of WT-Shc increased the insulin-induced association of SHIP2 with Shc, whereas a decrease in the amount of Shc on expression of **antisense** Shc mRNA led to a reduction in the SHIP2-Shc association. Furthermore, the inhibitory effect on insulin-induced Akt phosphorylation by WT-SHIP2 was decreased in **antisense**-Shc cells. These results indicate that the membrane

localization of SHIP2 with its 5'-phosphatase activity is required for negative regulation of insulin-induced Akt phosphorylation and that the localization is regulated, at least in part, by the association of SHIP2 with Shc in Rat1 fibroblasts.

L5 ANSWER 8 OF 13 MEDLINE on STN DUPLICATE 2  
 ACCESSION NUMBER: 2002054078 MEDLINE  
 DOCUMENT NUMBER: PubMed ID: 11694542  
 TITLE: Molecular events associated with CD4-mediated  
 Down-regulation of LFA-1-dependent adhesion.  
 AUTHOR: Mazerolles Fabienne; Barbat Christiane; Trucy Maylis;  
 Kolanus Waldemar; Fischer Alain  
 CORPORATE SOURCE: INSERM U 429, Bat. Kirmisson, Hopital Necker-Enfants  
 Malades, 149 rue de Sevres, 75743 Paris Cedex 15, France..  
 mazerol@necker.fr  
 SOURCE: Journal of biological chemistry, (2002 Jan 11) 277 (2)  
 1276-83.  
 Journal code: 2985121R. ISSN: 0021-9258.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 200202  
 ENTRY DATE: Entered STN: 20020125  
 Last Updated on STN: 20030105  
 Entered Medline: 20020207

AB We have previously shown that CD4 ligand binding inhibits LFA-1-dependent adhesion between CD4+ T cells and B cells in a p56(lck)- and phosphatidylinositol 3-kinase (PI3-kinase)-dependent manner. In this work, downstream events associated with adhesion inhibition have been investigated. By using HUT78 T cell lines, CD4 ligands were shown to induce a dissociation of LFA-1 from cytohesin, a cytoplasmic protein known to bind LFA-1 and to enhance the affinity/avidity of LFA-1 for its ligand ICAM-1. A dissociation of PI3-kinase from cytohesin is also observed. In parallel, we have found that CD4 ligand binding induced a redistribution of PI3-kinase and of the tyrosine phosphatase SHP-2 to the membrane and induced a transient formation of protein interactions including PI3-kinase; an adaptor protein, Gab2; SHP-2; and a SH2 domain-containing inositol phosphatase, **SHIP**. By using **antisense** oligonucleotides or transfection of transdominant mutants, down-regulation of adhesion was shown to require the Gab2/PI3-kinase association and the expression of **SHIP** and SHP-2. We therefore propose that CD4 ligands, by inducing these molecular associations, lead to sustained local high levels of D-3 phospholipids and possibly regulate the cytohesin/LFA-1 association.

L5 ANSWER 9 OF 13 CA COPYRIGHT 2004 ACS on STN  
 ACCESSION NUMBER: 135:317478 CA  
 TITLE: Enhancement of antibody-mediated immune responses by  
 disrupting FcγRIIB-mediated signaling  
 INVENTOR(S): Ravetch, Jeffrey V.  
 PATENT ASSIGNEE(S): Rockefeller University, USA  
 SOURCE: PCT Int. Appl., 46 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001079299	A1	20011025	WO 2001-US12106	20010413
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM,				

HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS,  
 LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO,  
 RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ,  
 VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM  
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,  
 DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,  
 BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG  
 US 2001036459 A1 20011101 US 2001-834321 20010413  
 EP 1272526 A1 20030108 EP 2001-926962 20010413  
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,  
 IE, SI, LT, LV, FI, RO, MK, CY, AL, TR  
 JP 2003531149 T2 20031021 JP 2001-576893 20010413  
 PRIORITY APPLN. INFO.: US 2000-198550P P 20000413  
 US 2000-204254P P 20000515  
 WO 2001-US12106 W 20010413

AB The present invention is related to enhancing the function of anti-tumor antibodies by regulating FcγRIIB-mediated activity. In particular, disrupting **SHIP** activation by FcγRIIB enhances cytotoxicity elicited by a therapeutic antibody in vivo in a human. The invention further provides an antibody, e.g., an anti-tumor antibody, with a variant Fc region that results in binding of the antibody to FcγRIIB with reduced affinity. A variety of transgenic mouse models demonstrate that the inhibiting FcγRIIB mol. is a potent regulator of cytotoxicity in vivo.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 10 OF 13 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN

ACCESSION NUMBER: 2001:559469 SCISEARCH

THE GENUINE ARTICLE: 451LN

TITLE: PTP-PEST, a scaffold protein tyrosine phosphatase, negatively regulates lymphocyte activation by targeting a unique set of substrates

AUTHOR: Davidson D; Veillette A (Reprint)

CORPORATE SOURCE: Inst Rech Clin Montreal, Oncol Mol Lab, 110 Pine Ave W, Montreal, PQ H2W 1R7, Canada (Reprint); Inst Rech Clin Montreal, Oncol Mol Lab, Montreal, PQ H2W 1R7, Canada; McGill Univ, McGill Canc Ctr, Montreal, PQ H3G 1Y6, Canada; McGill Univ, Dept Med, Montreal, PQ H3G 1Y6, Canada; McGill Univ, Dept Biochem, Montreal, PQ H3G 1Y6, Canada

COUNTRY OF AUTHOR: Canada

SOURCE: EMBO JOURNAL, (2 JUL 2001) Vol. 20, No. 13, pp. 3414-3426.  
 Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND.  
 ISSN: 0261-4189.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 59

\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*

AB There is increasing interest in elucidating the mechanisms involved in the negative regulation of lymphocyte activation. Herein, we show that the cytosolic protein tyrosine phosphatase PTP-PEST is expressed abundantly in a wide variety of haemopoietic cell types, including B cells and T cells. In a model B-cell line, PTP-PEST was found to be constitutively associated with several signalling molecules, including She, paxillin, Csk and Gas. The interaction between She and PTP-PEST was augmented further by antigen receptor stimulation. Overexpression studies, **antisense** experiments and structure-function analyses provided evidence that PTP-PEST is an efficient negative regulator of lymphocyte activation. This function correlated with the ability of PTP-PEST to induce dephosphorylation of She, Pyk2, Fak and Gas, and inactivate the Ras pathway. Taken together, these data suggest that PTP-PEST is a novel and unique component of the inhibitory signalling machinery in lymphocytes.

L5 ANSWER 11 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 3

ACCESSION NUMBER: 2000:360149 BIOSIS  
DOCUMENT NUMBER: PREV200000360149  
TITLE: **Antisense** modulation of **Ship-2**  
expression.  
AUTHOR(S): Bennett, C. Frank [Inventor]; Cowsert, Lex M. [Inventor]  
CORPORATE SOURCE: ASSIGNEE: Isis Pharmaceuticals Inc.  
PATENT INFORMATION: US 6025198 February 15, 2000  
SOURCE: Official Gazette of the United States Patent and Trademark  
Office Patents, (Feb. 15, 2000) Vol. 1231, No. 3. e-file.  
CODEN: OGUPE7. ISSN: 0098-1133.  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
ENTRY DATE: Entered STN: 23 Aug 2000  
Last Updated on STN: 8 Jan 2002

AB **Antisense** compounds, compositions and methods are provided for  
modulating the expression of **Ship-2**. The compositions comprise  
**antisense** compounds, particularly **antisense**  
oligonucleotides, targeted to nucleic acids encoding **Ship-2**.  
Methods of using these compounds for modulation of **Ship-2**  
expression and for treatment of diseases associated with expression of  
**Ship-2** are provided.

L5 ANSWER 12 OF 13 MEDLINE on STN DUPLICATE 4

ACCESSION NUMBER: 2000459945 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 10958682  
TITLE: 5' phospholipid phosphatase **SHIP-2** causes protein  
kinase B inactivation and cell cycle arrest in glioblastoma  
cells.  
AUTHOR: Taylor V; Wong M; Brandts C; Reilly L; Dean N M; Cowsert L  
M; Moodie S; Stokoe D  
CORPORATE SOURCE: Cancer Research Institute, University of California, San  
Francisco 94115, USA.  
CONTRACT NUMBER: R01CA79548 (NCI)  
SOURCE: Molecular and cellular biology, (2000 Sep) 20 (18) 6860-71.  
Journal code: 8109087. ISSN: 0270-7306.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200009  
ENTRY DATE: Entered STN: 20001005  
Last Updated on STN: 20001005  
Entered Medline: 20000922

AB The tumor suppressor protein PTEN is mutated in glioblastoma multiform  
brain tumors, resulting in deregulated signaling through the  
phosphoinositide 3-kinase (PI3K)-protein kinase B (PKB) pathway, which is  
critical for maintaining proliferation and survival. We have examined the  
relative roles of the two major phospholipid products of PI3K activity,  
phosphatidylinositol 3,4-bisphosphate [PtdIns(3,4)P2] and  
phosphatidylinositol 3,4,5-trisphosphate [PtdIns(3,4,5)P3], in the  
regulation of PKB activity in glioblastoma cells containing high levels of  
both of these lipids due to defective PTEN expression. Reexpression of  
PTEN or treatment with the PI3K inhibitor LY294002 abolished the levels of  
both PtdIns(3, 4)P2 and PtdIns(3,4,5)P3, reduced phosphorylation of PKB on  
Thr308 and Ser473, and inhibited PKB activity. Overexpression of  
**SHIP-2** abolished the levels of PtdIns(3,4,5)P3, whereas  
PtdIns(3,4)P2 levels remained high. However, PKB phosphorylation and  
activity were reduced to the same extent as they were with PTEN  
expression. PTEN and **SHIP-2** also significantly decreased the  
amount of PKB associated with cell membranes. Reduction of **SHIP**  
-2 levels using **antisense** oligonucleotides increased PKB

activity. **SHIP-2** became tyrosine phosphorylated following stimulation by growth factors, but this did not significantly alter its phosphatase activity or ability to antagonize PKB activation. Finally we found that **SHIP-2**, like PTEN, caused a potent cell cycle arrest in G(1) in glioblastoma cells, which is associated with an increase in the stability of expression of the cell cycle inhibitor p27(KIP1). Our results suggest that **SHIP-2** plays a negative role in regulating the PI3K-PKB pathway.

L5 ANSWER 13 OF 13 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 ACCESSION NUMBER: 1998:44397 BIOSIS  
 DOCUMENT NUMBER: PREV199800044397  
 TITLE: Keratin 8 and 18 expression in mesenchymal progenitor cells of regenerating limbs is associated with cell proliferation and differentiation.  
 AUTHOR(S): Corcoran, Jonathan P.; Ferretti, Patrizia [Reprint author]  
 CORPORATE SOURCE: Dev. Biol. Unit, Inst. Child Health, UCL, 30 Guilford St., London WC1N 1EH, UK  
 SOURCE: Developmental Dynamics, (Dec., 1997) Vol. 210, No. 4, pp. 355-370. print.  
 CODEN: DEDYEI. ISSN: 1058-8388.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English  
 OTHER SOURCE: Genbank-136454  
 ENTRY DATE: Entered STN: 27 Jan 1998  
 Last Updated on STN: 27 Jan 1998

AB Keratins are considered markers of epithelial differentiation. In lower vertebrates, however, immunoreactivity for keratin 8 and 18 has been reported in nonepithelial cells, particularly in mesenchymal progenitor cells of regenerating complex body structures. To confirm that such reactivity does indeed reflect keratin expression and to investigate their possible role in regeneration, we have isolated clones coding for the new homologues of keratin 8 and 18 (NvK8 and NvK18, respectively) and studied their distribution and changes in their expression following experimental manipulations. Analysis of NvK8 and NvK18 transcripts confirms that KS and K18 are expressed in the blastemal cells of regenerating newt limbs and that their expression is first observed 3-5 days after amputation, when the blastemal cells start to proliferate under the influence of the nerve, whose presence is essential for regeneration to proceed. In contrast, no induction of these keratins is observed following amputation of a larval limb at a stage when organogenesis is proceeding in a nerve-independent manner. To establish whether there is a causal relation. **ship** between keratin expression and cell proliferation in the adult limb blastema, we have investigated whether their expression is nerve-dependent and whether suppression of their expression in cultured blastemal cells affects cell division and differentiation. Analysis of keratins in denervated limbs demonstrates that the nerve is not necessary to induce their expression. However, treatment of cultured blastemal cells with KS and K18 **anti-sense** oligonucleotides significantly decreases DNA synthesis and induces changes in cell morphology, suggesting that expression of these keratins during regeneration may be necessary for the maintenance of the undifferentiated and proliferative state of blastemal cells.

=> d his

(FILE 'HOME' ENTERED AT 15:17:55 ON 22 JUN 2004)

FILE 'MEDLINE, BIOSIS, EMBASE, CA, SCISEARCH' ENTERED AT 15:18:01 ON 22 JUN 2004

L1 32068 S (SHIP (N) 1) OR (SH2 (2N) PHOSPHATIDYLINOSITOL PHOSPHATASE) O  
 L2 120052 S ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMEN (2N) (OLIGONUCL?  
 L3 140133 S ANTISENSE OR (ANTI (N) SENSE) OR (COMPLEMEN? (2N) (OLIGONUCL?

L4 26 S L1 AND L3  
 L5 13 DUP REM L4 (13 DUPLICATES REMOVED)  
 L6 3 S L5 AND PY<=2000  
 L7 0 S L4 AND (L3 (W) L1)

=> s Freier, S?/au; s Bennett, C?/au  
 L8 797 FREIER, S?/AU

L9 6026 BENNETT, C?/AU

=> s l9 or l8  
 L10 6788 L9 OR L8

=> s l10 and l1  
 L11 4 L10 AND L1

=> dup rem l11  
 PROCESSING COMPLETED FOR L11  
 L12 3 DUP REM L11 (1 DUPLICATE REMOVED)

=> d l12 ibib abs 1-3

L12 ANSWER 1 OF 3 CA COPYRIGHT 2004 ACS on STN  
 ACCESSION NUMBER: 139:47146 CA  
 TITLE: Antisense modulation of SH2-containing inositol  
 5-phosphatase (**SHIP-1**) expression  
 for treatment of inflammatory disorders  
 INVENTOR(S): **Bennett, C. Frank; Freier, Susan M.**  
 PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., USA  
 SOURCE: U.S. Pat. Appl. Publ., 46 pp.  
 CODEN: USXXCO  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003114401	A1	20030619	US 2001-3919	20011206
WO 2003053341	A2	20030703	WO 2002-US38622	20021204
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

PRIORITY APPLN. INFO.: US 2001-3919 A 20011206  
 AB Antisense compds., compns. and methods are provided for modulating the  
 expression of **Ship-1**. The compns. comprise antisense  
 compds., particularly antisense oligonucleotides, targeted to nucleic  
 acids encoding **Ship-1**. Methods of using these compds.  
 for modulation of **Ship-1** expression and for treatment  
 of diseases associated with expression of **Ship-1** are  
 provided.

L12 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 1  
 ACCESSION NUMBER: 2000:360149 BIOSIS  
 DOCUMENT NUMBER: PREV200000360149

TITLE: Antisense modulation of **Ship-2** expression.  
 AUTHOR(S): **Bennett, C. Frank** [Inventor]; Cowsert, Lex M. [Inventor]  
 CORPORATE SOURCE: ASSIGNEE: Isis Pharmaceuticals Inc.  
 PATENT INFORMATION: US 6025198 February 15, 2000  
 SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Feb. 15, 2000) Vol. 1231, No. 3. e-file.  
 CODEN: OGUPE7. ISSN: 0098-1133.  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 23 Aug 2000  
 Last Updated on STN: 8 Jan 2002

AB Antisense compounds, compositions and methods are provided for modulating the expression of **Ship-2**. The compositions comprise antisense compounds, particularly antisense oligonucleotides, targeted to nucleic acids encoding **Ship-2**. Methods of using these compounds for modulation of **Ship-2** expression and for treatment of diseases associated with expression of **Ship-2** are provided.

L12 ANSWER 3 OF 3 CA COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 121:110923 CA  
 TITLE: Manufacture and uses of biaxially oriented film comprising layers of polyethylene naphthalate bibenzoate  
 INVENTOR(S): **Bennett, Cynthia**; Kuhmann, Bodo; Ward, Bennett; Choe, E-won; Flint, John Anthony  
 PATENT ASSIGNEE(S): Hoechst A.-G., Germany  
 SOURCE: Eur. Pat. Appl., 20 pp.  
 CODEN: EPXXDW  
 DOCUMENT TYPE: Patent  
 LANGUAGE: German  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 580093	A1	19940126	EP 1993-111488	19930717
EP 580093	B1	19980729		
R: BE, DE, ES, FR, GB, IT, LU, NL				
DE 4224161	A1	19940127	DE 1992-4224161	19920722
DE 4238128	A1	19940519	DE 1992-4238128	19921112
JP 06199999	A2	19940719	JP 1993-180352	19930721
US 5919536	A	19990706	US 1996-630928	19960405
PRIORITY APPLN. INFO.:			DE 1992-4224161	19920722
			DE 1992-4238128	19921112
			US 1993-127891	19930721

AB The title film having increased stiffness and heat resistance, useful as a condenser dielec., a sail material, packaging or parting material, video tape substrate, etc., comprises  $\geq 1$  layer of a copolyester the acid component of which contains  $\geq 25\%$  of p-COC<sub>6</sub>H<sub>4</sub>C<sub>6</sub>H<sub>4</sub>CO units. Thus, an 8- $\mu$ m-thick title film of a di-Me 2,6-naphthalate-di-Me 4,4'-biphenyldicarboxylate-ethylene glycol copolymer (m. 281°) (preparation given) had elastic modulus 9.2 GPa, tensile strength 237 MPa, and elongation at break 25% in the longitudinal direction and 8.0, 182, and 17, resp., in the transverse direction.

=>